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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Sources of Error In Neurological Diagnosis

JOSEPH A. LUHAN, M.D.
Chicago, Illinois

I wish to make a few remarks in a spirit of contrition concerning an increasingly important segment of neurological practice—whether private or institutional—in the performance of which the myth of our omniscience is time and again exploded. I have been privileged to attend certain neurological conferences at a university medical college in this area wherein a wide variety of clinical and pathological material is presented, and have seen more than one case of obvious neurological dysfunction in which the diagnosis was a little hazy even with benefit of electroencephalography, spinal puncture, angiography, air studies and about every present day laboratory diagnostic aid short of measurement of selective uptake by different portions of intracranial substance of radioactive isotopes. The battery of experts convening at these meetings included persons of first rate attainment in neurology, neurosurgery and ancillary fields. Yet a distinguished service professor at this school once confided to me albeit facetiously that he rarely obtained any practical help from this galaxy of talent in the diagnosis of the more baffling cases. Furthermore, some of us can recall clinico-pathological conferences, such as those held at the Cook County Hospital, where the clinician discussing the case does not know the necropsy findings. Frequently after a distinguished clinician has made a diagnosis based on a learned and logical review of the evidence, the pathologist in gruesome triumph unveils an entirely unsuspected disease process, often with respect both to the site and nature of the lesions found. Do not be contemptuous of the poor pathologist: for is he not a doctor of medicine, and hath he too not ego? Seeing disease as he does in the light of hindsight the pathologist must envision clinicians with pretentions of diagnostic infallibility as foolish mortals indeed.

One may on sound grounds argue that diagnosis is only of academic importance in those cases in which the actual state of affairs can be verified by necropsy and the illness was unmodifiable in the light of our present knowledge. But there are a good many instances, as in the case of subdural hematoma, where an incorrect diagnosis may lead to an unnecessarily fatal issue. Furthermore, certain trends in private practice accentuate the need for the neurologist to become at least as good a diagnostician as he is a therapist and counselor. Within recent years some of our neurological statesmen have lamented the gradual constriction of the sphere of neurological practice wrought in part by advances in medical knowledge and the increase in specialization. Neurosyphilis waning to near extinction has been appropriated by the public health institutes and syphilologists, most organic brain diseases recognizable vaguely as such by the family physician are shunted to the enterprising neurosurgeons, cerebrovascular accidents have become the province of the internist, rehabilitation of the hemiplegic is

Presidential address read before a meeting of the Chicago Neurological Society.

Clinical professor of neurology and psychiatry, Stritch School of Medicine, Loyola University, Chicago.

performed by physiotherapists, poliomyelitis from its inception has been appropriated by the orthopedic surgeon, nearly all neurologic disorders of childhood are managed by the pediatrician and the more remunerative cases of psychoneurosis are sent to the psychoanalyst.

It is fortunate that the practicing neurologist, who is regarded as nearly therapeutically impotent by his colleagues, is concerned not only with museum cases but is still respected for his knowledge and is often called by the general practitioner to make a diagnosis, not only in frankly neurological disorders, but sometimes also in situations wherein the illness assumes a generally baffling nature. This implied compliment on the part of the family physician to call in the neurologist when no one else can make a diagnosis may stem from his impressions gained in medical school that neurological diagnosis is regularly feasible with mathematical exactitude to the initiated, and that when routine laboratory tests fail, the disease may be neurological or so called psychosomatic. For the purpose of overwhelming my patients and thereby easing their relinquishment of a fee for something so intangible as an opinion, I frequently explain how all this bludgeoning with a hammer and sticking with pins is part of an engineering feat to determine if and where transmission lines have been snapped. But alas! the answer to the question, "Where is the lesion?" is usually not so simple in actual private practice. Exquisitely focal lesions in the mine run of private patients implicating important centers or pathways are relatively rare; multiple lesions are common, and we are not dealing with monkeys suffering from the effects of experimental ablation of area 4, but with human beings possessing complex and often disordered personalities.

Some of the most elementary feats of neurologic localizing diagnosis impress the referring doctor as being spectacular accomplishments. Take for example the Wallenberg syndrome produced by occlusion of the posterior inferior cerebellar artery, whose anatomical basis is shown in Figure 1. The constellation of symptoms produced by such lesion is unique, rendering localization easy given a cooperative patient, and its vascular causation should be obvious from a reliable history. Recently, however, cases of metastatic tumor in this vicinity have been reported in which the neurologic deficit was like that found in the much commoner vascular lesion in this situation.

Figure 1. Cavitation in medulla oblongata from ancient softening in the distribution of one posterior inferior cerebellar artery; gross specimen.

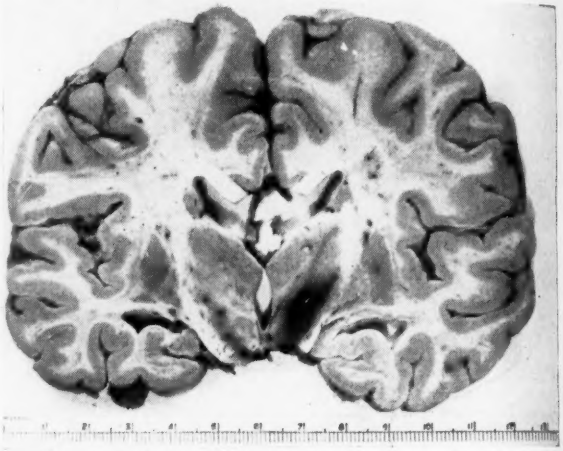

Figure 2 (a). Discrete hemorrhage in the subthalamic nucleus, causing the syndrome of hemiballismus.

Figure 2 (b). Weil stained preparations of comparable regions thru the basal ganglia of the two sides of the brain, showing a discrete hemorrhage in the right subthalamic nucleus (on the side of the artefact caused by a linear cut through the thalamus). The structures on the left side, for comparison, are normal.

Another type of spectacular disorder, ordinarily easy of localizing diagnosis, is the condition known as hemiballismus. Figure 2 shows a discrete hemorrhage into the right subthalamic nucleus complicating hypertension and cerebral arteriosclerosis in a 73 year old man who while in the act of shaving developed incessant, uncontrollable wild thrashing and flinging movements of the left extremities. Once in a while, however, no lesions are found in the subthalamic nucleus or its immediate connections, so that what might be expected to be perfectly simple with respect to localization turns out to be difficult, since the pathways interrupted in hemiballismus are complex.

Classical instances of sources of error in localizing diagnosis include the false localizing sign of abducens palsy in brain tumor, homolateral hemiplegia in intracranial space occupying lesions, frontal lobe tumor mimicking cerebellar disease, parasagittal cerebral lesions simulating spinal cord disease, and pontomedullary glioma in children imitating diffuse cerebral inflammatory disease. However, difficult as neurologic localizing diagnosis may be in many instances, the problem of etiologic diagnosis is often less susceptible of solution and of more practical importance.

An astute British physician, Ralph Winnington Leftwich, in the introduction to his book, "An Index of Symptoms"* wrote that diagnosis was the most difficult part of medicine, and that to the equipment of the ideal diagnostician something resembling legal acumen should be added so that statements by the patient and his friends might be sifted until only the truth remained. He went on to describe some of the fallacies of diagnosis. Following the examples of Leftwich and with a few additions, I have compiled a list with illustrations of some of the commoner sources of error in neurological diagnosis, apart from the group of false localizing signs previously alluded to.

1) *The Fallacy of Inadequate Examination.*

As a newcomer on the attending staff at Cook County Hospital, I examined a patient without benefit of interne. This patient was a middle-aged foreign born man who often drank alcoholic liquor to excess and one morning awakened to find his right arm limp and useless. I had not been aware that a famous professor had stopped here a few hours before with his entire entourage of internes, assorted residents, visiting physicians and medical students, and after a cursory examination and acceptance of the interne's findings, learnedly discoursed on Saturday night palsy as the nature of the patient's disability. The arm was flaccidly paralyzed, without deep reflexes. However, I noticed absence of the right abdominal reflexes and right homonymous hemianopsic field defects overlooked by the interne and concluded that the paralysis was cerebral in origin, an interpretation confirmed by subsequent developments.

The diagnostic consultant in neurology should beware of that feeling of superiority which makes him believe he can either dispense with the neurological examination or accept instead the findings of an interne. On the other hand examination with use of special diagnostic procedures can be carried out to impractical lengths and without avail in many cases. There can be no instrumental substitute for common sense. We can do harm to our private patients by untempered zeal in the advocacy of neurosurgical diagnostic procedures, or commit sins of omission by over-conservatism in our diagnostic efforts, just as some surgeons are too radical and others too conservative in their approach to surgical problems.

I recall my professor of urology at medical school, sitting before his class as if half asleep, droning on interminably about when to use and when not to use instruments, as if a urologist called in consultation could refrain from giving the patient "the works." Now I realize that this professor had been discussing something of great importance, applicable to any specialized diagnostic endeavor.

2) *The Fallacy of Variations Within the Normal*

A whole book could be written on this. I had recently examined in a nurses' infirmary a young graduate presenting headache and

*7th Ed. revised by H. N. Warner Collins, Wm. Wood & Co., N. Y., 1920.

slight elevation of the optic discs, who was found on further study to have an actinomycotic granuloma of the brain. Not long afterward in this same infirmary I examined another nurse, who complained also of severe headaches and who presented only slight elevation of the optic discs, the latter finding being confirmed by a distinguished ophthalmologist. The patient was referred as a brain tumor suspect to the Illinois Neuropsychiatric Institute. Normal spinal fluid findings were obtained, whereupon the patient was dismissed without air studies by a conservative neurosurgeon. Several years later this young lady, who had remained well, informed me that her brother while in service had been hospitalized and studied by a number of Army consultants because of peculiar fundoscopic appearances, and was diagnosed as having a normal abnormality (probably hereditary pseudopapilledema).

Once I examined a woman who had shattered her right squamous temporal bone with a bullet from a .22 caliber target pistol in a suicide attempt. She seemed none the worse neurologically for this experience, but I noticed that the knee and ankle jerks contralateral to the wound were greatly increased. However, perusal of a record of her hospitalization a year before in a different hospital revealed that a physician had observed the same difference in deep reflexes. Hence inequality in reflexes had no significance with respect to the bullet wound.

3) *The Fallacy of Atypical Onset or Course of the Illness.*

J. V., a man of 50 suddenly became nauseated, dizzy and had difficulty in maintaining balance while returning to Chicago on a train. He had been deaf in the right ear for several years. A year and a half previously he had a pigmented mole removed from the left forehead. Seen in my office he presented only slight instability of gait and was presumed to have a cerebellar vascular lesion, but was advised to enter a hospital for study to rule out acoustic neurinoma, among other things. X-ray films of the skull showed nothing abnormal, and spinal fluid findings were negative except for protein content of 89 mgm./100 cc. The patient became suddenly worse after caloric vestibular tests which produced normal nystagmus and past pointing, but caused excessive nausea and vomiting. He had to lie with his left ear on the pillow, and the slightest turning of the head provoked nausea and reflex vomiting; finally pylorospasm developed so that for a month he had to be nourished parenterally. Bilateral ataxia of the upper extremities with some intention tremor in the left was found soon after the Bruns' syndrome developed, but otherwise the findings were not remarkable; the optic discs, in particular were normal. I now suspected the presence of a cerebellar lesion, probably metastatic melanoma in the roof of the fourth ventricle, an opinion concurred in by a neurosurgeon. At the request of the family an older and more experienced neurologist was called, who after careful examination concluded that a cerebellar lesion, either vascular or neoplastic, was present, but because of the onset in the form of two sudden episodes and from intuitional bias leaned toward the diagnosis of a cerebellar vascular accident. Soon thereafter, the patient was able to retain fluids and food by mouth, became less nauseated, and could move his head carefully without vomiting. He was now able to sit up in a chair and seemed generally better. One month after his first examination the senior consultant noted marked improvement and neurologic findings compatible with residuals of a vascular accident in the brain stem. About three months after he first became sick the patient awakened one morning with motor aphasia and right sided facio--lingual-brachial weakness. This apoplectiform new development now impressed me as being vascular in origin, possibly on the basis of disseminated cerebellocerebral angiitis obliterans, although secondary vascular occlusion following growth of a nodule of metastatic melanoma had to be considered. The patient again improved in strength and ability to speak until he was discharged about three weeks after the aphasic episode. When he returned to the hospital in a dying condition two months later the cat was out of the bag: there were demonstrable pulmonary and cutaneous metastases. Necropsy revealed a mid-line cerebellar melanomatous tumor and other metastases in the frontal and parietal cortex on

the left side (Figures 3 & 4). The onset and clinical course, at the start like a series of apoplectiform episodes led to error in diagnosis.

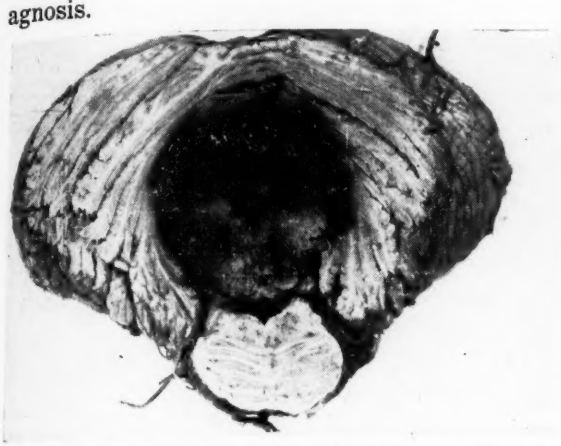

Figure 3. Case of J. V., showing a metastatic midline cerebellar melanomatous tumor.

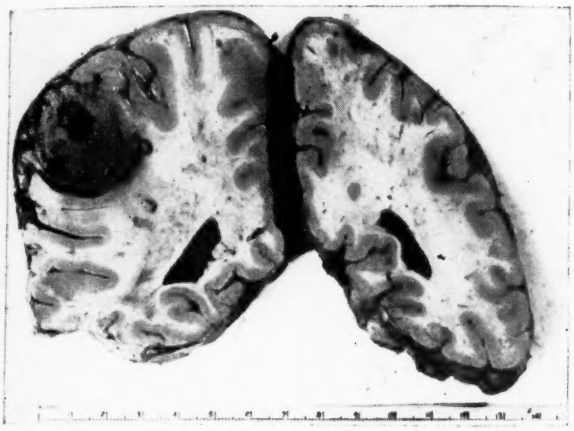

Figure 4. Case of J. V., showing a metastatic melanomatous nodule in the left parietal cortex.

4) *The Fallacy Created by Functional Overlay.*

It has been said that in its incipiency the name for multiple sclerosis is hysteria. Most of us are familiar with instances of multiple sclerosis in which a prior diagnosis of hysteria had been made. Conversely organic disease may be simulated by hysteria.

As an interne, I had under my care a middle aged man admitted for treatment of presumed renal colic: we found that his pain could be controlled by sterile water hypodermic injections, and that he was anesthetic for all cutaneous stimuli between the groin and the knee, typical of hysteria. A few months later this man was seen on the fracture service; X-ray had eventually revealed destruction of the bone surrounding an upper lumbar intervertebral foramen on the side of his pain consistent with metastatic carcinoma, and now that the nerve root was severed by the growth and no longer a source of pain, the objective sensory loss was in the conventional distribution of the affected lumbar root, instead of globally involving the thigh.

5) *The Fallacy of Antecedent Disease.*

A 43 year old white man was admitted to the hospital with a clinical picture resembling that of early tabes dorsalis, complaining of shooting pains in the legs, and showing absent Achilles jerks and incomplete left third nerve palsy. Five years before he had acquired a chancre and had 1½ years of antisyphilitic therapy. The diagnosis at first was syphilitic sacral posterior radiculitis and meningoneuritic oculomotor palsy. However, the clinical course finally led to the diagnosis of metastatic tumor. Chest X-ray on admission had been negative. The spinal fluid on initial puncture revealed normal findings except for 75 mgm. of protein per 100 cc. Three weeks after admission multiple cranial nerve paralyses had appeared; the knee jerks slowly disappeared, and motor as well as sensory involvement became apparent in the lower extremities, particularly in structures innervated from sacral segments. Ten weeks after admission the spinal fluid protein had

Figure 5. Metastatic carcinomatous nodules in the roots of the cauda equina.

Figure 6. Higher magnification of the same cauda equina specimen as in figure 5.

risen to 350 mgm., two weeks later still to 1300 mgm. per 100 cc. Necropsy revealed a bronchogenic primary source of metastatic carcinomatous nodules in the nerve roots of the cauda equina and at the points of exit of the cranial nerves. (Figures 5 & 6.)

6) *The Fallacy of Concurrent Disease.*

Figure 7 shows a sphenoid ridge meningioma compressing the basilar portion of the prefrontal lobe and the tip of the temporal lobe, in a 52 year old woman with serologically and pathologically demonstrable concomitant neurosyphilis: the focal symptoms were erroneously explained on a syphilitic basis, particularly in the absence of signs of increased intracranial pressure.

Figure 7. Sphenoidal ridge meningioma compressing the basilar portion of the prefrontal lobe and the tip of the temporal lobe on the right side, in a 52 year old woman with concomitant neurosyphilis.

Dr. Loren W. Avery, former attending neurologist at Cook County Hospital, used to say that syphilis was the poor man's privilege, and that syphilis was often mistakenly accused of being the cause of some other concomitant neurologic disease.

7) *The Fallacy of Laboratory Error.*

A 60 year old, heavily tattooed precinct captain of a flop-house district in Chicago began to exhibit irrational behavior. His physician, suspecting general paresis, drew blood for a Kahn test which was reported from the State Laboratories as being 4+. By the time the patient was admitted to a private psychiatric unit he was wildly disturbed and had to be restrained. His blood pressure was normal. Blood and spinal fluid

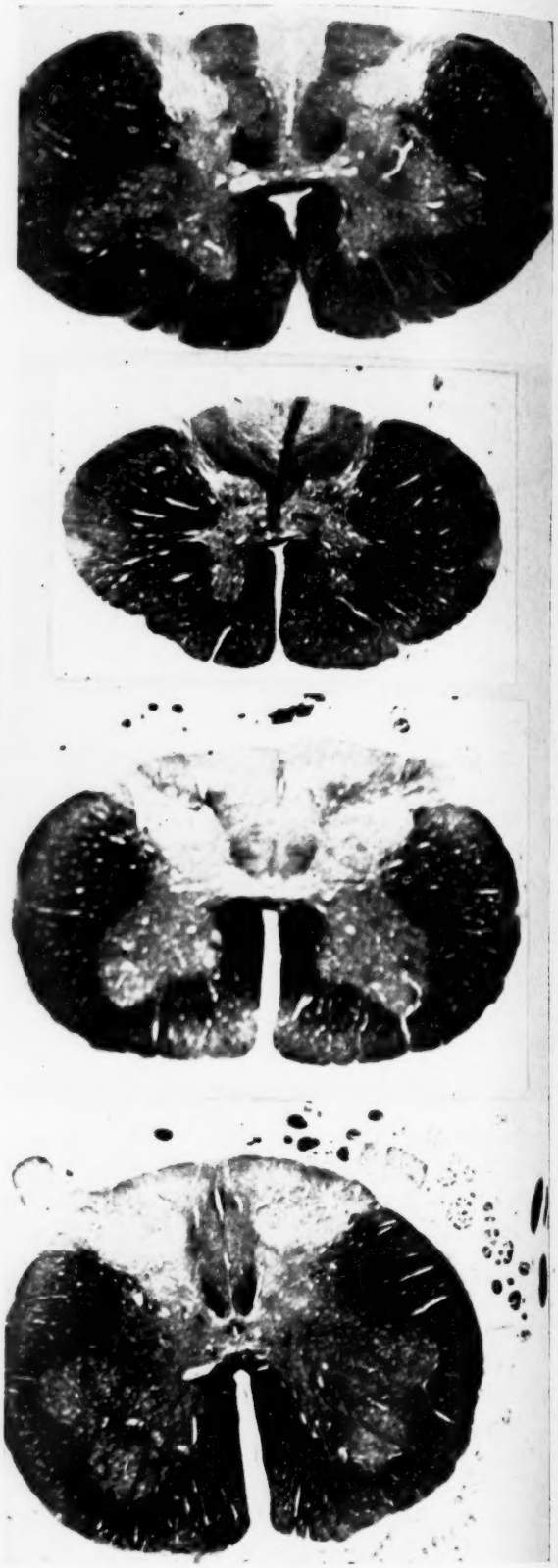

Figure 8. Posterior column degeneration clinically masked by coexistence of a severe peripheral neuropathy; sections thru the cervical, thoracic, upper lumbar, and sacral cord segments stained for myelin by the Weil method.

tests for syphilis done at the hospital were negative, but careful history taking from relatives disclosed that a former physician had long ago prescribed some medicine for "blood pressure." A high blood thiocyanate level was found. The patient soon recovered from his toxic psychosis caused by thiocyanate intoxication.

8) *The Fallacy of Neutralization or Overlapping.*

Recently the case of a young man who had suffered from diarrhea for years and became bedridden because of severe peripheral neuropathy, was the subject of a clinicopathological conference. The pathologists in this case differed in their diagnosis and interpretations of the disease process which clinically seemed to be the result of idiopathic steatorrhea. A finding in the spinal cord, which could not be detected during life because of the neutralizing or masking effect of the peripheral neuropathy, was that of a severe degeneration in the posterior white columns of the cord (Figure 8).

9) *The Fallacy of the Diagnosis of What is the Lesion? being Influenced by Where is the Lesion?*

A 55 year old white woman who after a fall developed spastic weakness of the legs and later of the arms with atrophy of the small hand muscles, without sensory or sphincteric disturbance; she died ten months after onset of her illness, which was diagnosed as amyotrophic lateral sclerosis. Necropsy revealed that the disease was multiple sclerosis and that the oldest and most severe lesions were in the cervical region, involving the lateral white and the anterior gray columns (Figure 9).

Other diagnostic fallacies include:

10) *The Fallacy of Post Hoc Ergo Propter Hoc*

For example, necropsy may reveal that death following a blow on the head had been due to cerebral embolism.

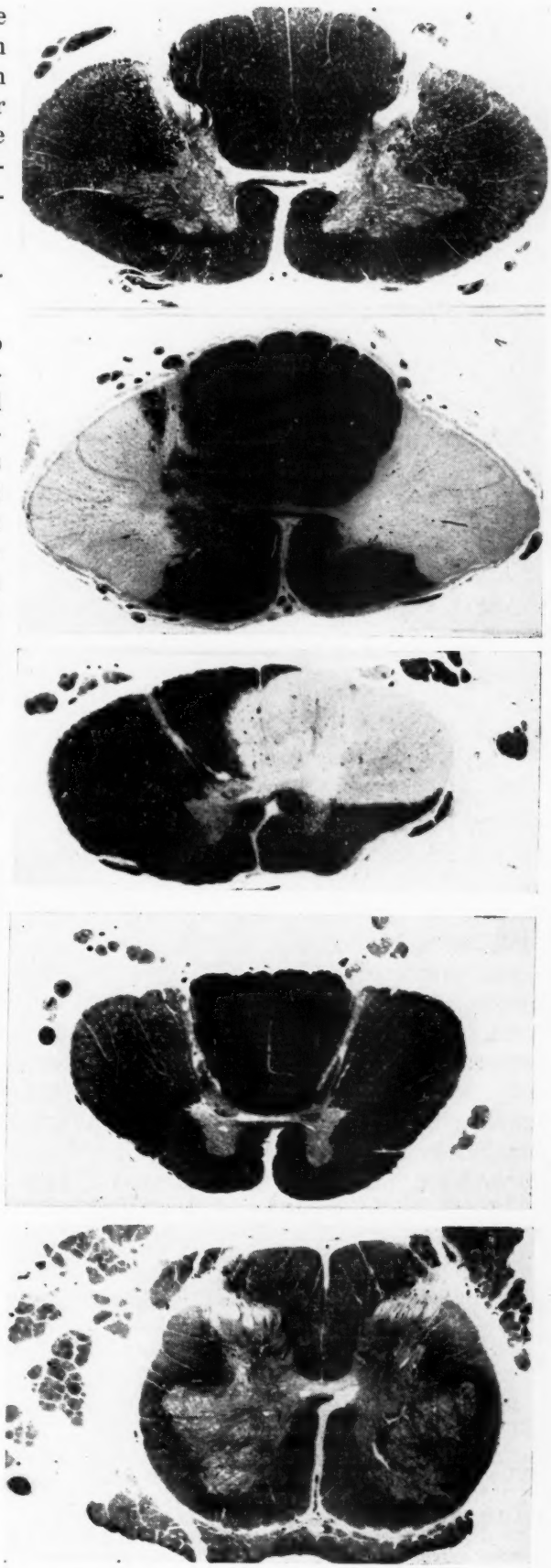

Figure 9. Amyotrophic form of multiple sclerosis. These spinal cord sections, stained for myelin by the Pal-Kultschitzky method, from above downward are thru the fourth cervical, eighth cervical, third thoracic, eighth thoracic, and first sacral segments, respectively.

11) *The Fallacy of the Pathognomonic Sign.*

There are very few absolutely pathognomonic signs in medicine. I have seen an otherwise perfectly typical case of myasthenia gravis in which prostigmine, used for the first time with this patient, failed to influence the muscular weakness.

12) *The Fallacy of Perspective on the Part of the Examining Physician.*

The junior consultant often errs in constantly expecting to unearth a very rare disease.

13) *The Fallacy Introduced by Desire for Compensation or by Malingering.*

This is known to all consultants dealing with veterans and in medicolegal cases.

14) *The Fallacy of Suggestion.*

The physician may create symptoms in neurotic individuals and be misled by such iatrogenic disease.

This is the type of record of errors and human failings committed by well informed clinicians, as can be attested by internes and residents who seem to know more than their teachers and who in turn will commit such errors themselves. I can well understand, therefore, the need in competitive private practice for the creation by the distinguished neurological consultant of a myth of infallibility, as even a first rate star of the cinema can use a little press agentry for the enhancement of glamor and box-office appeal.

29 E. MADISON ST.

School Experience in Training of the Child Psychiatric

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Training in child psychiatry, characteristically, by its emphasis on pathological behavior, frequently fails to give the psychiatrist in training an opportunity to observe normal children. It also frequently fails to familiarize him with contemporary educational processes. As part of their training program in child psychiatry the authors spent one day each week as observers at the Ethical Culture Midtown School in New York City. This experiment in expanding the training program for child psychiatrists was started in 1949. The original plans were formulated jointly by Dr. S. Bernard Wortis, Professor and Chairman of the Department of Psychiatry, New York University College of Medicine, and Mrs. Victoria Wagner, Principal of the Ethical Culture Midtown School. During the training period an average of three or four days was spent with each grade level, from the nursery through sixth grade. The remainder of the training program was devoted to the diagnosis and treatment of emotionally disturbed children in hospital settings, including out-patient clinics, and to research and teaching of medical students.

One of the principal objects of the study at the school was to observe the developmental processes of children. We were able to see years of child development unfold before our eyes in a few months as we progressed from grade to grade. Norms of development could be observed more readily and more accurately in groups, where the extremes are balanced, than by observation of isolated pathological children in the clinics. In these circumstances it was relatively easy to witness the unfolding of the child's personality as he matures and explores his world and discovers techniques for dealing with it. The transition from the narcissistic, pleasure-striving child of three to the more reality-oriented twelve-year-old who is consciously working for a variety of goals could be clearly seen by watching children in the successive school years.

From the Department of Psychiatry, New York University College of Medicine.

The Ethical Culture Schools were founded by Dr. Felix Adler. They have evolved out of what began in 1878 as a free kindergarten. They are nonsectarian, progressive schools, whose enrolment reflects the diversified population of New York City, and whose curriculum emphasizes the importance of the individual in his relation to society.

Inseparable from the individual development of the child is the change in the development of his social relationships. Here, too, the transitions are striking and were clearly demonstrated at the school. The three-year-old, almost completely individualistic, who even when apparently playing with another child, is really merely playing alongside him, is followed by the four-year-old who makes the first attempts at group play, though the group rarely lasts more than a few minutes. By five or six, the child is capable of organized games and a modicum of collaborative work, and by eight, complex group activities are the rule. It is necessary for the psychiatrist to have a picture of the social capacities of normal children in order to evaluate the complaints of parents and the play patterns of disturbed children.

One of the interesting aspects of this experience was the opportunity to observe children of a different socio-economic group from that customarily seen in clinic experience. The children at the school revealed essentially the same problems and character traits as do those seen in clinics, but their manifestations are determined by characteristics of their social and cultural group. Sibling rivalry was more likely to be manifested by competition about clothes or intellectual eminence, rather than in direct aggression. In addition one frequently saw the nurse-reared child whose parents, though present, were secondary figures in the child's life. There was also a greater sophistication in the school children which was not simply a function of their intellectual superiority but largely a product of cultural influences and also of more intensive and richer verbal associations with adults than is common with lower-income group children. On the other hand, one saw the influence of certain factors in American cultural life spreading across class and economic lines—particularly those of television and the Hopalong Cassidy fad, which are as prominent in the Park Avenue children as in those of the lower East Side. Thus, both contrasts and similarities can be noted; it is the value of this experience that we had the chance to make comparisons.

We also had an opportunity to obtain a panorama of the educational process in action through the elementary grades. Like any educational system, progressive education is as much a function of the teacher as of the educational theory, and the variations in educational technique at the school gave testimony to the imagination and experimental interest of the faculty. It was rewarding to witness an educational process based on the needs of the child and directed toward encouraging full development of his creativity. Indeed, it was this last point that was most impressive to us. The range and the level of creative activity among the children in the school were, of course, a function of the culture from which these children come, and of their generally superior intellectual capacities, but the freedom given them by the school permitted maximum development of these creative capacities in a way which few children reared in a more restrictive educational system can match.

In addition, it was revealing to note the variations in educational approach with the increasing maturational level of the child, and thus the awareness and grasp on the part of teachers of the developmental process. The value of the collaboration between psychological research, psychiatry, and education was amply demonstrated in progressive educational techniques such as we have seen here.

By having the opportunity to observe and participate in the classroom activities, we also gained an increased awareness of some of the problems in the relationship between teacher and child. The teacher's role is significant not only in terms of her relationship with the group but also in terms of the particular experiences of the individual child. We were able to observe this interaction and to appreciate in a more meaningful way the problems teachers face in attempting to meet the needs of the child. Each child brings into the school situation attitudes patterned in terms of his earlier relationships at home and structures the school situation for himself in this way. The teacher's personality enters into this relationship, and this, too, influences the unique meaning of the school experience for the individual child.

Although our interest was not focused on the problems of deviant behavior of certain children, this was seen as an integral part

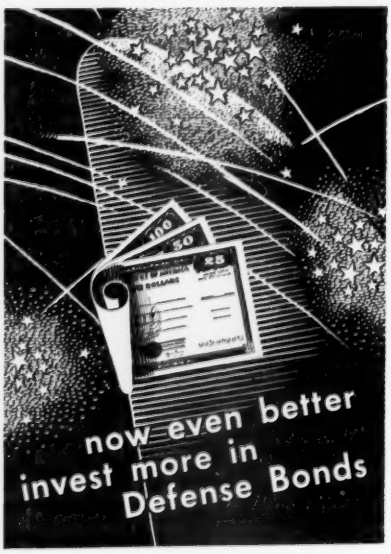
of the total school situation. We were able to observe to what extent the school could serve as a positive experience for the child and in so doing increase the potentiality for his healthy emotional development. That certain behavior patterns in children were not accessible to the environmental setting the school provided was apparent, and we had the opportunity to observe how psychiatric facilities were integrated into the functioning of the school in meeting the needs of these particular children, and how important a close working relationship between the parent, the school, and the psychiatrist is in working out a successful program for the treatment of the emotionally disturbed child.

It was valuable for us to recognize the role the school plays in helping parents to accept psychiatric treatment and to realize how much preliminary planning must be done by the school before a child is referred for psychotherapy. In this connection we gained an increased awareness of the relationship of the school to the community needs. The school plays a definite role in disseminating information to parents and in serving as a liaison between the psychiatrist and the child.

Conclusion

Active participation in the daily classroom experiences as an adjunct to the more usual methods of training of child psychiatrists has proved to be of great interest and value. It has brought the psychiatrist into close contact with a major area of children's experiences that until now has been seen only indirectly. The authors have had the opportunity to observe normal children in their normal habitat, to become familiar with the problems that arise in the educational process, and to know something of the experiences and difficulties of teachers and administrators. This program may represent the first step in the formation of a closer liaison between psychiatrists and educators which will prove of mutual benefit.

We know that our experience has been atypical in that the school involved was one with unusual facilities and one which draws from an upper socio-economic group, and that limitations of time restricted our opportunity for more thorough observation. Nonetheless, we feel that this pilot study may prove of value in pointing the way for more extensive programs utilizing the facilities of public schools and psychiatric training centers.



Psychotherapy in Medical and Surgical Hospitals

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A discussion of psychotherapy for patients in a general hospital must, of necessity, include matters that are beyond the powers of observation and techniques of non-psychiatrists and must deal with unconscious processes. These are not necessarily limited to forces operating only within the patient, but include transactional operations with the surrounding human environment—doctors, nurses, attendants, relatives, etc., and demands emanating from the institutionalized social roles that characterize being a patient, being hospitalized, etc. Thus a discussion of psychotherapy extends from the patient to his human and physical environment and is psychiatric only insofar as its methods assist the observer in understanding the dynamic interplay among the variables of patient, sickness and environment.

In our society man is constantly called upon to play a variable number of differentiated roles from which both he and those appropriate and stereotyped behavior.mong with whom he interacts expect a relatively appropriate and stereotyped behavior. Among these roles, that of being ill is experienced at sometime by almost everyone and by some with greater frequency and longer duration. In this role a patient is forced to give up his characteristic activities, his freedom from direction and to relinquish his active role to play a passive obedient part in response to the advice and commands of his physician. Giving up obligations, responsibility and mastery, the sick person instead appeals for help, encouragement and support. In so doing he becomes dependent to a greater degree than his ordinary adult pride and his usual social environment permit. He regresses to some degree of dependency with appropriate inactivity, to a state of physical quiescence of his whole being which has the same reparative value as immobilization and rest to a sick part. The strains responsible for this primary disintegrative process and the strains in the organism inherent in the adjustive processes which are expressed as disease are accompanied by general fatigue and weakness due to the focalization of energy demands and require psychological and physical rest.

The need for regression and rest from activity and responsibility varies with individuals and cannot be scheduled. Often the therapeutic ambitions of the physician for rapid miraculous cures are obstructed by a slow recuperative process causing frustration within the doctor and subsequent impatient pushing of the patient. This only intensifies the regressive pull within the individual patient who has his own rate of recovery, as each child with his own rate of development, resists and reacts against pressure. I have been called upon many times to deal with the "neurosis" of slow recovery and found that lessening pressure alone made for more rapid recovery.

In our hospitals today it is not as much the rationality of preventing weakness or circulatory inactivity but rather the shortage of beds and dearth of nurses that are responsible for the pushing of patients through short convalescence. Psychologically this is as absurd as infrequent psychotherapeutic sessions to prevent dependency. One should as soon advise fewer hours of sleep as preventive for liking sleep too much. An appropriate time for dependency, and sleep, and convalescence are all necessary for recovery from illness. However, the extent of time and the degree of necessary dependency are often exceeded when reliance for support on the magic of the authoritative omnipotent physician-priest-father or the gentle loving care of the giving mother may fulfill unconsciously wished-for satisfaction. In these patients the unconscious or secondary gain of illness may create a new system of adjustment for which the physician unwittingly has been given a role as a causative or perpetuating factor. Complaining bitterly and creating new symptoms as they are needed, the recovering patient

From the Institute for Psychosomatic and Psychiatric Research and Training of the Michael Reese Hospital.

finally becomes a trial to the physician who then asks for psychiatric help. These patients cannot be treated in a hospital setting with its emphasis on tests, physical and pharmacological therapies but should be discharged and deterred from seeking further diagnostic or medical procedures to enable psychotherapy to proceed. Often the simple technique of clarification and explanation accompanied by exploration of overstraining life situations are helpful if at the same time the anxious physician is also adequately dealt with psychotherapeutically.

Another type of patient refuses to give up control and mastery and cannot accept the role of patient in need of help. He denies illness and refuses immobilization. He worries about work, business or profession and attempts to carry these on by telephone, correspondence or conferences. It is this type that demands complete information of every step in diagnosis and therapy and attempts to be part doctor in consultative capacity with his own physician. To be "strong and doing" is his motto and often he thereby jeopardizes his own life. Patients with coronary thrombosis and some with peptic ulcers often fall into this category. In general they can be best handled not by force or persuasion, but by accepting them in the role of defiant and cooperating rebels against the demoniacal illness that creeps within them without their participation. Others can only be dealt with by interpretation of "sickness as an undesirable weakness" as a frustration of their habitual overcompensation. The fight against the need for love and security as a general pattern can be seen as the background to the fight against illness. The therapeutic gain is usually only temporary but sometimes enough to tide the patient over the period in which activity is most dangerous.

The role of the physician is not as stereotyped as it once was when embodied in the family physician who subscribed to the Hippocratic oath and was an intuitive practitioner of the healing art for body and soul. Today the attending physician on a hospital staff is highly specialized, very busy and a worshipper of the American factory "work-up" production system. He embodies many types of personalities yet he still is concerned about his patient's welfare for one reason or another and becomes anxious when frustrated in diagnosis, angry (often at the patient) when unsuccessful in therapy and given to vacillate or to compromise between gentle understanding and pompous domination. Since his threshold of consultation for the patient's good or for the record is low the psychiatrist is being called into consultation with greater frequency and liaison psychiatric services have educated the general physician or specialist about the psychiatrists' functions and how they can be utilized to best advantage.

Some physicians are dedicated as of old to the gentle art of healing and others to the competitive life of narcissistic achievement. It is the latter who react with anxiety to failures of diagnostic procedures and physical or pharmacological therapies and need guidance and treatment from the psychiatrist.

The need of the physician for psychotherapy is in fact the most important concern of the liaison psychiatrist. One may question whether I should not have expressed this as education instead of therapy. The latter word was chosen because learning by explanation and demonstration is an endlessly repetitive and, for the most part, unsuccessful process. The physician can only learn through experiences in which he participates emotionally and that means allaying his anxieties through uncovering his specific roles and sensitive spots in patient-doctor relationships as they have become part of his general life pattern. Sometimes this is accomplished by frequent frank discussions regarding special patients and in a few instances the doctor is stimulated to further introspection or analysis. However the result can well be the stimulation of great resistance and hostility mobilized against the psychiatrist and psychiatry unless the subject for such a process is carefully chosen.

The institutions of hospital, nursing and food service are beyond the control of individual patients, doctors or psychiatrists. They are rigid and firmly structuralized and far behind advances in medicine and humanism. Time of awakening, feeding and bathing, medication rituals and a host of other procedures can only be altered by spe-

cial order for each individual patient. The significance of these routines for the patient as it effects his progress is rarely applied by the physician; that becomes a function of the psychiatrist who is concerned with the patient and his stresses.

Psychiatrists are often called to see patients who develop confusional states, paranoid ideas and verbalizations of suicide intent when recovering from serious physical illness. Although conscious for many hours of the day during their illness, ego functions have been carried on automatically with little expenditure of energy most of which has been utilized in the somatic reparative processes. The return of active ego functions often occurs with the suddenness of a startled awakening from sleep with no gradual transition period. The confusion, the misidentifications and the distortion of reality as though it were a dream state make the patient appear psychotic. It has been my experience that decreased activity in the patient's sick room, the presence of the most reliable, closest relative who calmly and slowly unfolds the happenings of the missed days or weeks along with mild sedation, serves to bring the patient's ego to a recognition of the world about him.

In this connection I should like to mention the elderly patient who is often hospitalized for some upper respiratory or other mild infection. At night he may be found wandering in the halls in a dazed state or observed attempting to open or jump out of a window. During the day he is less confused. Such a patient has little ego capacity to grasp the meaning of a suddenly shifted and unfamiliar world and searches for land-marks and the accustomed points of reference. All of us sleeping away from home on awakening suddenly, have looked around at the strange order of external things before grasping where we were. The elderly patient has much greater difficulty and slower capacity of adjustment. His confusion can be overcome only by sending him home to the old familiar human and physical environment or, if hospitalization is absolutely necessary, by keeping him asleep during the mysterious hours of darkness.

The usual order of events in modern medical diagnosis, especially in hospitalized cases, is for the physician to do a "work-up." This includes a variety of routine tests and some especially significant for the organ or system suspected to be in dysfunction. When all these have failed to disclose structural alterations or organ dysfunctions, the psychiatrist is invited in a last-chance hope for diagnosis. Most of these patients complain of intra-abdominal distress, headache, diarrhea, etc. It is not the absence of physical findings that is significant for the psychiatric diagnosis. The psychiatrist is interested in the positive evidence of emotional conflicts that may be significantly related to the presenting symptom at onset and during intensification or relapse and evidences of meaning in the language of the disturbed organ and in the absent verbal communications. Conflicts, emotional disturbances, overt neuroses, etc., are always present in varying degrees; it is their relation to dysfunction that leads to correct diagnosis and adequate therapy.

Sometimes psychiatric consultation is requested after a physical disturbance properly diagnosed and adequately labeled has been treated fruitlessly with standard procedures. The various consultants cannot understand why change does not occur and ward patients are discussed endlessly by troops of attending men and their assistants. Finally, emotional factors are considered as mitigating against or delaying recovery and the psychiatrist is consulted. For these cases I have been adamant that the patient be transferred to the psychiatric unit, the medical men forbidden access to the patient's bedside and the case assigned to a single psychiatrist under supervision.

Here are two examples:

Mr. B., a twenty-two year old asthmatic, had been hospitalized for months and slowly became worse. No medication seemed to stop his spells and the quantity of adrenalin compounds and derivatives used in twenty-four hours was enormous. He was in a constant state of asthma for which there was little relief available and he knew all the remedies tried by his physicians and all their doubts. He was transferred to the psychiatric unit and under the care of Dr. George Ham he improved rapidly and soon was free from all but minor attacks, leaving the hospital in four months. For the next two years he was treated by psychotherapy and gradually progressed to become independent enough to live away from home, work and enjoy the com-

pany of females. There were many relapses when meaningful life situations developed or when crucial points were reached in his therapy, but from the day of isolation from zealous physicians and the substitution of one consistent, dependable therapist he improved steadily.

A nineteen year old boy had developed diarrhea, nausea, abdominal cramps and occult blood in his stools at the age of seventeen after a long background of puny childhood. The medical diagnosis was terminal ileitis for which he had many intestinal operations and the removal of much diseased tissue. The genetic factors of patient-mother parasitic relationship need not be discussed except to indicate that they seemed of more signicance than any hypothetical bacterial or viral source of granulomatous tissue. After removal to the psychiatric unit under the entire care of Dr. Edward Frank the patient steadily improved and needed no further destructive surgery. There were many relapses in and out of hospital but in general the patient developed as much comfort as could be expected from a parasite living apart from maternal hostess.

Some patients admittedly coming under the responsibility of both internist and psychiatrist are handled with great difficulty during such a division of labor. A patient assigned to Dr. George Ham suffered from a syndrome diagnosed as hyperthroidism. During psychotherapy the thyroid disturbance increased in intensity when latent rage was stirred up by life situations and interpretations. During one of these periods the cooperating internists became alarmed and insisted on heroic medical measures to save the patient's heart from damage and preserve his life. It took many discussions and eventually a full-dress conference to allay their anxieties, and even then they privately considered the psychiatrists as criminal. The patient recovered and lived to be a statistic in a published report.

On the other hand a patient with ulcerative colitis was treated medically on the general ward and psychiatrically by a young psychiatric resident. The therapist had been warned about the possibility of a psychotic outcome which is often the fate of psychiatrically treated person with ulcerative colitis and became anxious and timid, focusing his attention on the symptoms. The patient became worse and released herself from the hospital. Shortly afterward she returned to the medical service where a confident and blunt medical resident took complete charge. He laid down positive rules of conduct and insisted on their fulfillment. No visitors were allowed, demands were curtailed; only he was in charge of the total person of the patient. With this firmness, by-passing the symptom and a determined therapeutic zeal which protected the patient from parents and nurses, the patient responded by gaining weight and left the hospital in a complete remission which has now lasted for months.

The most distressing request for the psychiatrist is the last minute frantic call before operation by the surgeon who suddenly becomes anxious over his intended intervention. A twenty-four year old veteran had been wounded in the knee in Europe and for many months was shunted from one army hospital to another. He was finally discharged with limited motion of the leg diagnosed as ankylosis and given a 50 percent disability pension. I arrived too late to see the patient who was already in the operating room being prepared for a tendon elongation. To the surgeon's surprise under anesthesia the leg was easily flexed. In chagrin he stopped the anesthesia and wrapped up the offending limb in a plaster cast which I insisted on having removed the next day. In a pentothal interview he revealed that while in combat he had discussed the subject of self-inflicted wounds with his buddies and debated with himself about the possibility of getting away with it. The enemy helped him make the difficult decision with a mild shrapnel wound and he was happy for a brief period. The guilt over desertion of buddies set in and the boy started his self-punitive career. At the end of the pentothal interview he found himself walking normally around the room with me as he regained consciousness. After another three days of psychotherapy the patient walked out of the hospital and has remained well for several years.

A thirty-nine year old man, a patient of Dr. Sidney Portis, had been suffering from severe abdominal pains in the right upper quadrant for two years and was sent to Chicago from West Virginia for an exploratory operation. The pain was severe, radiating to the back, worse at night and associated with nausea, vomiting, pallor, sweating and

writhing. A brief exploratory interview revealed a serious intrafamily conflict over business in which the patient culpably lost a considerable amount of his parents' money. The patient was sent home for psychotherapy which was effective without the necessity for exploratory operation to allay the anxiety of his local physician.

Another patient whom I had treated before because of many psychogenic bowel disturbances had been taken out of therapy by his father because he became too independent. After the lapse of a year, father and son had a bitter quarrel and the break between them seemed final. The patient developed signs of liver disease, diagnosed as infectious hepatitis. His condition seemed hopeless and heroic measures were contemplated as the liver was almost entirely nonfunctioning and jaundice was severe. At this point I was called back and recognized that the primary psychodynamic process had gone to the extreme of almost causing death. It was inadvisable to delay operation for a period of therapy so that I could only help in preparing him for the drainage of the bile ducts. He rallied emotionally in his relationship with me and faced the operation bravely. Fortunately this decompressed the stasis of hepatic bile and the patient lived. Subsequent therapy was continued until a new psychological equilibrium was reached but further psychoanalysis was delayed until full recovery because of the danger of stirring up more pathogenic rage toward the parents.

There are many unique situations in which each patient is an experiment in evaluation: For example, some patients reveal evidence of severe physical dysfunction, but with considerable and obvious emotional disturbances so that question is raised regarding their role in etiology. Dr. Winston Breslin has studied a few patients in whom he could affirm that depression and hopelessness were indications of the patient's unconscious awareness of the malignant nature of his illness before his medical attendants had arrived at that diagnosis. Some patients in defense of an intuitively garnered knowledge of malignancy develop a neurotic pattern which permits them to allay anxiety of impending death. This can also be induced therapeutically and is of value in saving the patient psychological pain of facing death.

I shall not do more than mention the fact that countless numbers of problems are so simple that any physician with common sense and the patience to listen for a few moments to the sick under his charge can deal with them adequately. Nor will I go into the frustrations of seeing patients who are not prepared at all for a psychiatric consultation or who are sensitized against the consultation by the bungling clarification of ambivalent attending physicians.

What is the function of the psychiatrist in the problems of acutely ill patients? He can't psychoanalyze the patient or even give him much psychotherapy in a busy ward, a two-bed room or even in a private room with attendants and relatives coming and going, especially when the patient is too sick to move. The miracle of a sudden knife-like incision into the unconscious by an omniscient psychoanalyst is for the moving pictures only; if it happens in real life it must be rare. On the other hand an ill-advised sentence, a misplaced jocularity or a serious statement wrongly emphasized can disintegrate the patient's recovery processes and result in a tragic outcome.

In acute disturbances our tasks are adequate diagnosis, understanding of the multiple interacting factors of patient, environment and doctors as they have been genetically related to the causes of the illness and as they relate in the present to the hastening or impedance of recovery. With such understanding we may prevent harm from impinging on the patient and encourage promising directions of recovery. Those forces which we can block or set into action, as the case may be, must be dealt with in the mind of the responsible physician from which radiation to relatives, nurses and the hospital ritualists may occur. Relationships with the patient directly which satisfy his need of the moment may facilitate his participation in the recovery process. However, when it comes to direct psychotherapeutic intervention the patient must be handled by the psychiatrist alone without interference, in privacy and with ample time.

Treatment of Behavior Disorders Incidental To Organic Cerebral Disease by Prefrontal Lobotomy

(Report of Seven Cases)

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Psychopathologic behavior disturbances due to acquired organic brain disease constitute one of the most difficult problems in psychiatric treatment. Not only are the usual therapeutic measures ineffectual as far as cure is concerned, but the actual problem of home or hospital management is difficult in the extreme. The hyperactivity and destructiveness of these patients constitute an almost insurmountably difficult nursing problem. Nothing short of complete isolation in locked rooms makes the hospital environment safe for many of these patients. The tendency to self-mutilation and uncontrollable rages makes the patients actively dangerous to themselves and others.

In the seven cases reported here the patients showed: Self-mutilation and concomitant arson, attacked attendants and physicians, (case 1); constant overactivity, necessitating tying to avoid escape and refusal to wear clothes, (case 2); destruction, clawing dangerously at mother's eyes and face, (case 3); arson and attempted homosexual rape, (case 4); serious self-biting, biting of others, assaultiveness, (case 5); destruction of household furniture and clothes, and constant screaming and running away, (case 6); constant overactivity, destructiveness of household furnishings and difficulty in management, (case 7).

Gibbs,[1] Hohman,[2] Hall[3] and Hill[4] have adequately described the behavior disorders seen in patients during and following the epidemic of encephalitis which began in 1919. These patients all showed extreme emotional lability characterized by tantrum-like outbursts of rage, which occurred at times with minor provocation but often without apparent cause. Frequently there was a history of restless hyperactivity with impulsiveness and constant and marked irritability. The concomitant association of certain psychopathic features, such as sexual perversions, erotic tendencies and criminal traits, added to the difficulty in controlling these patients. A review follow-up,[5] made one to nine years after the 1918 epidemic showed no abatement of the psychopathology in 58 out of 85 cases unless the patient developed severe Parkinsonism (2 cases). The chronicity of the disorder was also apparent in Gibb's[1] series, in which 84 of the original 144 children who had encephalitis were still in hospitals three or more years after the onset of their illness thus adding to the problem of long-term care of the patients.

The advent of prefrontal lobotomy and the success of this operation in controlling pathological emotional states and psychotic overactivity, such as that seen in chronic manic and schizophrenic states, led the authors to believe that the operation might be of benefit in the treatment of severe behavior disorders following acquired organic brain disease. The favorable report of Thorpe[6] in the treatment of two adult cases of post-encephalitic behavior disorder by lobotomy supported the authors' belief that the operation might be of value in younger patients who showed marked chronic behavior disorders following either epidemic or other types of encephalitis. The following is a report of seven cases:

Case 1, J. J.: Post-encephalitic behavior disorder. A 14-year-old white female, who in June, 1947, was first noted to be groggy and unsteady on leaving a theater. The following day she developed a ptosis of the left eye, was fidgety and became incontinent. She was admitted to Duke Hospital and extensive investigation, including viral studies, led to an exclusion diagnosis of Von Economo's lethargic encephalitis. The patient's symptoms were described as typical of the syndrome. The patient had some choreiform movements during the acute phase of

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her illness. Two months after being discharged from the hospital she showed delirioid symptoms, thinking that she was being poisoned. Eight months later the patient began to have attacks of uncontrollable rage. At this time, she was treated with benzedrine with no improvement. Eleven months after the onset of her illness the patient's attacks of rage had become longer in duration and more uncontrollable. On separate occasions the patient set fire, first to the house and then to the barn, burning both of them to the ground. Because of the parents' inability to control the patient, she was committed to the State Hospital at Raleigh. In the hospital, the patient continued to exhibit the same type of behavior. Once in a fit of rage, the patient ate several large pieces of glass. A gastrostomy was done, following which the patient developed a draining sinus in her abdomen, into which she would insert all types of foreign substances. Frequently the patient attacked physicians and attendants in the hospital. The pre-operative physical examination revealed the patient to be a large, hyperactive, white female, who was constantly moving. A Parkinson-like tremor in both hands was constant. The patient continuously kept tearing up a kerchief into small bits or kept knotting a string during the entire examination. Emotional control was absent. A rapid shift from laughing to crying was observed, and at times the patient would laugh and cry simultaneously. Neurological examination revealed the patient's reflexes to be equal but hyperactive. No rigidity or mask-like facies was observed.

The patient was lobotomized on Nov. 29, 1949 under endotracheal ether anesthesia. The post-operative course of the patient was uneventful, except for transient hallucinations of a delirioid nature.

Several weeks later there was a slight change in the patient's emotional reaction. It, however, remained extremely labile, but the aggressive attacks were no longer observed. The rages had become of short duration and usually did not terminate in self-mutilation or violence. Two months post-operative, the patient showed some tendency to return to her previous behavior pattern, and during this period on one occasion set fire to the mattress in her room. Since that time the patient has shown only minimal improvement, and at the present time is still markedly hyperactive, constantly tearing at strips of cloth, ripping her clothes as well as those of other patients. Although the patient is no longer as combative and assaultive as she was before the operation, there is little change in her condition.

Case 2, E. McN.: Post-encephalitic behavior disorder with mental deficiency. This six-year-old, white, female was in good health until the age of 15 months, when she developed an illness which was characterized by fever and a short convulsion. Twelve days after admission to the hospital, the patient developed an upper-respiratory infection, with a temperature of 105° F. She again had convulsions, with greater severity on the right side. Following the convulsions the patient had a mild hemiparesis, and remained semi-comatose for approximately a month. A lumbar puncture was done, which revealed an increased intraspinal pressure but no increase in cells or protein. Two months after the onset of the illness, the patient seemed retarded, but the right hemiparesis had begun to clear. A month later the only residual of the patient's illness was an almost imperceptible weakness on the right side and marked mental retardation. After recovering from her original illness, the patient suffered grand mal convulsions, approximately every six months. Following her illness no intellectual development was observed and she became animal-like in her behavior, constantly running, would tear clothes from her body, and could be controlled only with difficulty. At times, it was necessary to restrain the patient. Pre-operative physical examination revealed a markedly hyperactive, white, female child who required the restraint of two attendants. No spontaneous speech was observed and it was impossible to test her intellectual faculties. Neurological examination was negative. Physical development was normal. Intellectual development was estimated to be two years.

The patient was lobotomized on Aug. 22, 1949, under endotracheal ether anesthesia. Post-operatively the patient was much improved, and played quietly in her crib for three days. At the end of this time the patient once again began her hyperactivity, and this has continued unabated since. At the present time, the patient shows no change over her pre-operative state.

Case 3, S. M.: Post-encephalitic behavior disorder with epilepsy. This five-year-old white child was said to have developed normally except for convulsions at the age of three and six months, associated with acute febrile illnesses. At the age of 2½ years she contracted a cold with a series of convulsions. Several days after the onset of this illness, the patient developed swelling at the angel of the mandible, and during the ensuing period the convulsions became more frequent, often as many as 17 per day were observed. Also a gradual change in her behavior was noted. The previously normal mental development ceased and she became markedly over-active. Her speech became thick and although she had previously been bilingual she retained the ability to speak only one language. She was extremely destructive and would scratch and claw at her parents' eyes during the night. The acute episode then subsided, and by the age of four the convulsions gradually decreased in frequency, but her behavior remained essentially unchanged. A complete neurological survey, done at the age of four, was essentially negative. Medication with anti-convulsants affected only the patient's convulsive seizures.

When seen in October, 1949, psychometrics revealed the patient to have an I.Q. on Stanford-Binet of 58, with a mental age of 2 years 6 months. Examination at that time revealed a white female of

five years, who was constantly moving about the room and controlled only with difficulty. Her speech at periods lapsed into an unintelligible jargon and most of her coherent utterances were two to four-word combinations. Neurological examination was within normal limits. Electroencephalogram was normal. A diagnosis of post-encephalitic behavior disorder with epilepsy was made.

The patient was lobotomized Oct. 31, 1949, under endotracheal ether anesthesia. Post-operatively the patient was much improved for a brief period and her hyperactive behavior disappeared. However, following discharge from the hospital she relapsed and resumed her previous hyperactivity. Her aggressive behavior was decreased for one year but 2½ years later, the patient placed in a boarding school became uncontrollable and institutionalization was being considered by the parents. There is at this writing no significant change in the patient's general behavior.

Case 4, R. C.: Post-encephalitic Parkinsonism with psychosis. This patient was considered normal until the age of 21, when he developed psychotic episodes characterized by hallucinations, delusions of persecution and delusions of grandeur which persisted, and within six months the patient developed marked over-activity and destructiveness. After he set fire to a mattress he was committed to a State Hospital, where a diagnosis of schizophrenia was made. In the hospital the delusions persisted. Shortly after admission he began to develop a Parkinsonian tremor, more marked on the right side. Three years after admission to the hospital (age 24), the patient began to have oculogyric crises. In addition, the patient developed marked aggressive homosexual activity, as well as auto-erotic tendencies which were uncontrollable. His delusions and hallucinations persisted unchanged and after eight years (age 29) the patient's symptoms became stationary. Pre-operative physical examination revealed an emaciated, stooped, rigid, white male, whose facial expression did not change except when he attempted to talk. A marked Parkinson tremor involved the patient's right arm, with lesser involvement of the left. His eyes at the time of examination were rolled back in his head, and it was impossible for him to bring his eyes down enough to look at the examiner. His mouth was held widely open and a Parkinson-like tremor involving his tongue was observed. Cog-wheel rigidity was present in all extremities. Reflexes were hyperactive on the left and hypoactive on the right. His speech was intelligible. Numerous delusions of persecution and of grandeur were present. The patient believed that he was a German (false) and intimated that he wished to return to his native country. He also felt that people were trying to beat him, and also that voices were saying vulgar things about him.

The patient was lobotomized in March, 1951 (age 41) under endotracheal ether anesthesia. At the time of operation, marked gliosis was encountered in the patient's left frontal lobe. Post-operatively, he seemed better. His personal appearance improved and he showed none of his aggressive homosexual behavior. However, the patient made frequent escape attempts and continued to have hallucinations, delusions of persecution and ideas of grandeur. His auto-erotic activity persisted unabated and he remains at present in an institution. It is not considered that this patient has shown significant improvement other than a decrease in his aggressive homosexual behavior.

Case 5, E. F.: Cerebral palsy with behavior disorder and mental deficiency. This 21-year-old white female was admitted to the State Hospital from the State School for Mental Defectives because of the patient's hyperactivity, vulgarity, assaultiveness and combativeness. At the school for mental defectives, the patient had been involved in many altercations with other patients; she would attempt to claw at the faces of others; she had bitten several other patients severely and had been self-mutilative. No information was available regarding the patient's birth and early development and no adequate medical history was available.

Mental examination revealed an uncooperative, noisy, laughing, white female, who was observed to have a severe strabismus and a marked limp. Her speech was coherent and relevant and filled with many vulgar expressions. Physical examination revealed a moderate enlargement of the head with an indentation of the skull on the right, kyphosis of the spine with concavity to the right. An internal strabismus was noted on the right, and diminished reflexes were observed on the left, with weakness of all muscle groups on the left. A diagnosis of behavior disorder incidental to organic cerebral changes was made. Bilateral prefrontal lobotomy was carried out on February 14, 1950 under endotracheal ether anesthesia. At operation, the patient was found to have a severe hydrocephalus on the right, with less than 1.5 centimeters of cerebral tissue remaining intact. Post operatively, the patient was markedly improved. The biting episodes virtually disappeared and the patient's speech was less vulgar and her self-mutilation had stopped. The patient, however, remained institutionalized because of her mental deficiency. Two years later, however, the patient resumed her previous behavior.

Case 6, R. D. B.: Post-encephalitic behavior disorder with mental deficiency. This eight-year-old, white female was said to have developed normally until the age of six months. At this time, the patient had begun to sit up alone, but following an episode of flu, accompanied by fever and convulsions, the patient did not sit up again until she was a year old. Between the age of six and twelve months, the patient had little use of the right side of her body but gradually regained strength in her extremities. She walked at two years, but has never talked to any extent. Since age two, the patient has been excessively overactive, destructive, and has required constant surveillance to prevent her destructive acts. The neurological examination, which was done preoperatively, revealed no signifi-

cant abnormalities. A Gesell Developmental Rating, done at the age of four, revealed less than 18 months development. Because of her overactivity, it was impossible to test the patient prior to operation. Operation was undertaken at the request of the child's parents who wished to make the management of the child less difficult. This was preceding her commitment to a hospital for mental defectives. The parents hoped she would receive kinder care if she were less destructive. Bilateral prefrontal lobotomy was done under endotracheal ether anesthesia on August 1, 1951. The post-operative course was uneventful. Following the operation, the patient was improved. There was less overactivity and she showed no tendency to be noisy as she had been pre-operatively. A follow-up six months later revealed the patient to be essentially the same as she was pre-operatively. Management was said to be less difficult.

Case 7, W. A. S.: Behavior disorder incidental to cerebral trauma (?) Infectious(?) This six-year-old white male was originally seen because of his inability to talk and his violent and uncontrolable behavior. The patient's birth was essentially normal, and he walked at one year and was making sentences at the age of 18 months. At the age of three the patient had two serious falls, the latter of which resulted in unconsciousness after falling out of bed. At approximately the same time, the patient was said to have had an episode of measles which was not accompanied by unconsciousness or by convulsive seizures. Following this, the patient became unmanageable, destructive, thieving, attacked servants and ran impulsively into the path of automobiles. At the time of admission to the hospital, he showed unintelligible speech at times, and never more than echo-like utterances. The patient was destructive on the ward, grimaced constantly, was distractible and markedly overactive. A bilateral prefrontal lobotomy was done July 1, 1949, under endotracheal ether anesthesia. Three months later, the patient was slightly improved. Five months following his operation, he had relapsed to his previous aggressive, overactive behavior. At the end of 2½ years, the patient was only slightly improved, was still overactive, but there had been some decrease in his aggressive destructive behavior. It was felt that this patient was improved, but the persistence of destructiveness and restlessness makes intitutionalization necessary.

Comment:

Although the report of Thorpe[6] showed a marked improvement in two cases of post-encephalitic behavior disorder which were lobotomized, the patients of the authors showed no significant improvement. The hyperactivity which the patients displayed was not at all affected. It was concluded by other authors that the hyperactivity of these patients was not due to a mood change but was a motor hyperactivity due to organic cerebral changes.[7] This is in keeping with the results on all the patients in this series, since the hyperactivity did not seem to be affected by the operation, and emotional reactions were not sufficiently developed in the majority of these patients to have them modified by the operation. It is not considered that the treatment of these patients by lobotomy is valuable, mainly because the destructiveness and hyperactivity from which these patients suffered was not retarded. Although one patient who had been dangerous was no longer so, her status as a whole was not changed. No patient was significantly improved.

Summary and Conclusions:

1. Seven cases of behavior disorder incidental to organic brain damage were lobotomized.
2. None of the cases was improved.
3. A slight flattening of emotional response was observed in two patients with slight improvement in management.
4. Hyperactivity was not affected in any of the patients.
5. The results of prefrontal lobotomy for organic brain damage in seven cases has been poor.

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Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE NO. 272756

Presentation

A 60-year-old colored woman was admitted to the hospital on March 21, 1951, because of inability to walk and urinary retention. She had been in good health until early 1950 when she began to complain of weakness and fatigue. On June 10, 1950, while working as a maid, she went to stool and on attempting to rise, her left knee collapsed. She was unable to get up from the floor and her employer took her home. She had been bedridden since that time. During the period following June 10 she complained of such severe burning of her feet that even the bed clothes were uncomfortable. She said that she could move all of her extremities fairly well but that her legs would not support her. She had no visual impairment or difficulty in swallowing. She apparently had no convulsive seizures, though she spoke vaguely of loss of consciousness on several occasions. Considerable weight had been lost due to extreme anorexia. There was no abdominal or chest discomfort until the week before admission when she developed suprapubic pain and urinary retention. She had used alcohol liberally (exact amount unknown) prior to the onset of the present illness but denied its use since. She had been cared for at home by her mother who corroborated the history and stated that a physician had not been called because they expected that the patient would get well.

Examination on admission to the hospital revealed normal vital signs in a woman who was apparently chronically ill but in no acute distress. Brief mental examination indicated that she was oriented as to person and place but not time. She objected to questioning. There was great impairment in simple calculating. She was not sure who was president of the United States. She complained constantly, not wanting to be annoyed by examination.

On physical examination there was evidence of much loss of subcutaneous tissue. The function of the cranial nerves was normal. There was papillary atrophy of the tongue which was light red in color. She was able to move all extremities but strength was reduced throughout. Severe ataxia was present in the lower extremities on the heel-knee-shin test, and there was slight past-pointing without tremor in the upper extremities. Sensory examination was difficult to evaluate because of poor cooperation. There was apparent loss of the appreciation of vibration and of touch below the pelvis. Pain seemed impaired below the costal margin in that there was a considerable delay between stimulus and response. The deep reflexes were hypoactive in the upper extremities and absent at the lowers. Abdominal skin reflexes were absent. Plantar responses were extensor bilaterally.

Hematologic examination revealed 2.6 million red blood cells per cmm., hemoglobin 12.8., gm., 1.1 reticulocytes, 99 thousands platelets, 12,200 white blood cells. HCT was 38 MCV 145, MCH 45, MCHC 33. The differential white count showed 85 per cent polymorphonuclears, 13 per cent lymphocytes, 1 monocyte, and 1 myelocyte "C." The red blood cells were noted to be macrocytic with anisocytosis and poikilocytosis. Examination of the urine was normal save for a trace of albumin and a few microorganisms. Blood urea nitrogen was 56 mg. per 100 cc. Blood Wassermann reaction was negative. A lumbar puncture was done on March 26; the fluid contained total proteins of 57 mg. per 100 cc; Wassermann and colloidal gold curve were negative.

Roentgen examination of the lumbar spine was normal. On the day following admission she was found in moderate respiratory distress, apparently not using her intercostal muscles at all. Therefore, she was placed in a respirator where she remained for two days. She was treated with a gram of streptomycin daily, 200,000 units of penicillin thrice daily, and 15 micrograms of vitamin B_{12} twice daily. On March 25 it was noted that she was breathing well when out of the respirator and her chest was clear. On the morning of March 26 she suddenly went into shock and became comatose for no apparent reason. Despite heroic efforts at therapy, including two blood transfusions, she continued to go downhill and died that afternoon.

Differential Diagnosis

*Dr. Richard W. Vilter:** This was a 60-year-old colored woman who came into the hospital when it finally became obvious to her that the human mechanism would not fix itself. She gave it nine months' trial and it failed; a failure of sufficient degree to bring her problem to this conference. Her illness began with weakness and fatigue, and this was followed by anorexia, and finally she was unable to use her left leg. The onset was sudden and might suggest that she had some type of vascular accident, but actually the initial manifestation of a metabolic disorder may also occur suddenly. For example,

*Associate Professor of Medicine. Director of the Laboratory of Hematology and Nutrition.

I've seen patients with severe alcoholic neuritis whose first manifestation was inability to lift a beer glass to the mouth.

Our patient went to bed and then developed a series of symptoms and signs which have varied significance. In the first place, she had severe burning in the lower extremities. This symptom raises the possibility that she had peripheral neuritis principally of the sensory type at that time. Then she developed urinary retention, suggesting that her bladder was involved in the same neurologic process. Finally when she came to the hospital she had mental aberrations, slight confusion, and considerable irritability which I believe are manifestations of cerebral dysfunction in this instance.

On examination it was found that she had muscular weakness but not paralysis. There was ataxia in the lower extremities and to a questionable degree in the uppers. She had loss of vibration perception and of touch below the pelvis, and pain sensation apparently was diminished below the costal margin. There was some difference between the levels at which these sensations disappeared. She had areflexia in the lower extremities and extensor plantar responses. We can associate these various neurologic symptoms and signs with involvement of particular neurologic systems. The rather diffuse muscular weakness, the story of the hyperesthesia that she had at home in bed when it hurt so much to have the bed clothes touch her feet and legs, the ataxia, the areflexia, and the sensory loss in the lower extremities are all probably the result of peripheral neuritis in severe form. The ataxia and the vibratory perception loss in the lower extremities could be loss of proprioception from disease of the posterior columns. This possibility is strengthened by the story of difficulty in emptying the bladder while she was still at home, suggesting that the proprioceptive fibers in the wall of the bladder were involved also. The most logical level of such involvement is the posterior columns. This woman had an atonic bladder suggesting posterior column disorder rather than a spastic contracted bladder which would suggest a transecting lesion of the cord. The atonic bladder certainly fits with the other two manifestations which suggest posterior column disease, ataxia, and vibratory perception loss, but these two signs could be due to peripheral nerve involvement as well, and we are quite certain that she had peripheral neuritis. One way we might differentiate between peripheral nerve or posterior column disease would be to find vibratory perception diminution extending fairly high up the spine. Such a sign would be quite indicative of posterior column involvement rather than peripheral neuritis. One can make such differentiation on the basis of how much vibratory perception loss has occurred in the spinal column in comparison with how much involvement exists in the lower extremities. With much in the lower extremities and little over the spine one thinks of peripheral nerve involvement; with much over the spine and relatively less over the lower extremities one would think of posterior column involvement. In addition to that, she had extensor plantar responses indicative of pyramidal tract disease, and she had mental degenerative changes as indicated by her irritability and her confusion. She also had manifestations of systemic disease associated with this neurologic disorder. She had glossal atrophy, anemia of the macrocytic type, and thrombocytopenia. Her white count was not as depressed as I would expect it to be, probably as the result of an infected bladder. At least, she had infection some place and the bladder would be the most logical site. Ordinarily with this syndrome one would expect a reduction in the white count as well as in the platelets. Lumbar puncture showed normal fluid save for slight elevation in the protein which is not uncommon in any spinal cord degenerative disease.

Of course, in order to have been a perfect case, this patient should have had histamine refractory achlorhydria, a megaloblastic bone marrow, and an elevation of the indirect reacting bilirubin in the peripheral blood. That's all we would really need to complete the diagnostic features of pernicious anemia with severe peripheral neuritis, postero-lateral column degeneration, and cerebral degeneration The most common neurological manifestation is involvement of the peripheral nerves, which begins most often in the lower extremities; the posterior

columns are next most frequently involved; thirdly, the lateral columns; fourthly, the cortical areas with psychosis, with delusions, sometimes hallucinations, and when these occur they are most commonly of the paranoid type; and fifthly, cranial nerves also may be involved with loss of the sensations of smell and taste. Sometimes deeper structures in the brain may be involved causing nystagmus. I think it is fairly certain from this protocol that this woman did have pernicious anemia and a severe involvement of the peripheral and central nervous systems. If that be the case, then the ultimate cause for it all was lack of intrinsic factor (the gastric factor) in the gastric juice, with reduction in absorption or almost complete lack of absorption of vitamin B_{12} from the food. Because of this conditioned deficiency, abnormalities in the metabolism of the bone marrow, of the spinal cord, and of other tissues occurred.

There is one pertinent but difficult question which might be asked. Why did this woman have such severe neurologic degeneration with only moderate involvement of the bone marrow? That, from theoretical considerations, is not what one would expect if one considers this a deficiency state, a deficiency in vitamin B_{12}. The deficiency of an essential metabolite often causes dysfunction in several systems, but that system should be involved most severely which has the greatest metabolic turn-over. The bone marrow is much more active metabolically than the nervous system, so one would expect major bone marrow and peripheral blood involvement and less involvement of the nervous system. That is usually the case. Only 33 per cent of patients with pernicious anemia in the initial relapse have moderate or severe involvement of the nervous system though 66 per cent have minor signs of neurologic disease. With each relapse more and more symptoms and signs of severe nervous system involvement appear. Eventually most will have nervous system involvement if they go on long enough without treatment, but by and large it is usually blood first and nervous system afterwards. However, one does see this type of case and sometimes the preponderance of nervous system involvement is even more striking. I have never seen a proved case of pernicious anemia where nervous system involvement occurred years or even months before blood involvement. Where such diagnoses has been made in the past, eventually the patient turned out to have multiple sclerosis or something of that nature. I do not believe that it is possible to have spontaneous untreated pernicious anemia manifested only by involvement of the nervous system. One may have mild anemia and severe neuropathy, however. How can this happen? There are a number of possibilities. In the first place one might guess that even though this woman was relying on God and nature to take care of her illness, still the vitamin advertisements had made an impression and she sent her mother to the drug store to get some vitamins for her, and these vitamins contained a certain amount of folic acid. If she had done this, the folic acid might well have prevented severe hematologic involvement, and the neurologic disease might have progressed without hindrance because folic acid does not benefit these neurologic manifestations. As a matter of fact, continued treatment with folic acid under these circumstances further depletes the body of what little vitamin B_{12} it has left and may lead to exaggerated nervous system involvement.

The second possibility is that the patient represents a rare dicotomy between the metabolism of folic acid and vitamin B_{12}. Let us reconstruct such a situation: A patient, because of intrinsic factor deficiency, is unable to absorb vitamin B_{12} adequately from the gastroenteric tract, but nothing goes wrong at all with the absorption of folic acid or with the metabolism of folic acid. Most hematologists now believe that in pernicious anemia the deficiency of vitamin B_{12} in some way adversely effects either the absorption or the metabolism of folic acid, so that a combined deficiency of both vitamins occurs. Should the patient continue to get adequate folic acid from the food and metabolize it correctly, the blood count would be maintained at a value slightly lower than normal, but the neurologic manifestations would go on unchecked or might be exaggerated.

Thirdly, previous alcoholism might have damaged this patient's peripheral nerves and

spinal cord so badly that she couldn't withstand a moderate deficiency in vitamin B_{12}; or the alcoholism may have induced sufficient additional deficiency disease such as a deficiency in thiamin, that a combined deficiency of B_{12} and thiamin had a more deleterious effect on her peripheral nerves and spinal cord than a single deficiency might have had.

Even though this is such a cut and dried proposition we ought to consider other possibilities in differential diagnosis. What are other causes of postero-lateral column degeneration and why do they not pertain here? In the older age groups one occasionally sees patients with advanced arteriosclerosis involving the peripheral nerves and the spinal cord, producing a syndrome which is indistinguishable from postero-lateral column degeneration of pernicious anemia. It may go on through all the various manifestations described here and may ultimately end in death from bladder infection. Under these circumstances, however, one usually does not have macrocytic anemia, and the neurologic degeneration is a rather slow, progressive affair; it doesn't progress rapidly as in this particular instance. Achlorhydria will not help very much because in the age group in which arteriosclerotic postero-lateral column degeneration occurs, the majority of patients have achlorhydria anyway whether they have pernicious anemia or not. The anemia and the glossal change rule out arteriosclerosis as an etiologic factor in this case. In the younger age groups when this type of syndrome occurs, one must always think of multiple sclerosis, and as far as I know most of those patients previously thought to have the neurologic manifestations of pernicious anemia without the anemia, have turned out eventually to have multiple sclerosis. Neither arteriosclerosis nor multiple sclerosis, of course, will respond at all to refined liver extract or vitamin B_{12} whereas with the postero-lateral column degeneration of pernicious anemia one would expect slow improvement over a period of 6 to 12 months, or at least no progression of the disease. I should mention also that there are idiopathic cases of postero-lateral column degeneration.

Other types of neurologic involvement which have to be thought of are the various neuritides. This woman was a user of alcohol, so one would wonder whether alcoholic neuritis might possibly explain her clinical picture. Very serious involvement of peripheral nerves, cord, and brain may occur in such patients. This has been called central neuritis. However, in order to relate the anemia to this etiology, one would have to say that it was due to a dietary deficiency in both folic acid and vitamin B_{12} and this would be most unusual without severe manifestations of other vitamin B complex deficiency states, or that it was due to a severe degree of chronic liver disease with macrocytosis. The latter is unlikely because one seldom sees such high mean corpuscular volumes in the macrocytic anemia of chronic liver diseases. It was 145 in this case. Nor have I seen such progressive and severe neurologic signs for so long after the patient is said to have stopped the alcohol intake. Such signs may last for six months, but then they gradually improve if the alcohol has been stopped and the patient begins to eat again.

Other forms of neuritis and neuropathy should be mentioned, but they scarcely warrant consideration. There is the neuropathy of lead poisoning, arsenic poisoning, bismuth poisoning, and following the ingestion of Jamaica ginger. The last might be considered because this woman had consumed alcohol and Jamaica ginger paralysis may occur in alcoholics who would use this drink when they could get nothing else. It produces not only signs of peripheral neuropathy but also of cord disease with extensor plantar responses, spasticity, and ataxia. However, again the anemia and rapid determination cannot be explained on the basis of "Jake Paralysis."

Other causes of neuropathy such as mixed porphyruria and infectious neuronitis must be considered but are most unlikely. The age of the patient and the absence of gastroenteric disturbances, particularly constipation, argue against the first possibility. The spinal fluid protein is against the latter.

Tabes should be considered in this patient. After all, she did have posterior column involvement, areflexia in the lower extremities, and atonic bladder. However, her spinal fluid Wassermann and gold curve were nor-

mal, and this progressive disease can scarcely be called burned-out tabes where one might find a normal cerebrospinal fluid. Furthermore, the pupils and discs were normal, she had normal vision, and she had no symptoms in the past suggesting tabetic crisis.

Finally, some of her manifestations, particularly the sensory levels that are suggested at the costal margin and at the pelvis, might point to a transverse lesion of the cord. Such a lesion might occur in conjunction with anemia if caused by involvement of the vertebrae with either lymphoma or metastatic tumor. However, under those circumstances the anemia would not be macrocytic and x-rays of the spine would have showed a lesion in the bone as the origin of such cord compression.

We are left with the only diagnosis that I think is at all reasonable, postero-lateral column degeneration, and degenerative changes in the cortex as a result of pernicious anemia which in turn is due to a deficiency in vitamin B_{12}.

Dr. Charles D. Aring: Dr. Vilter, as I remember, you and I saw a patient a year or two ago, an employee of this institution, who had a tremendous cerebellar syndrome, and, if I recall correctly, she had what was considered to be a relatively normal peripheral blood, but a bone marrow quite typical of pernicious anemia. Is this impression correct? Does this combination of factors ever occur? If so, it would explain what we see occasionally in the literature, that neurological signs with some frequency precede changes in the blood, that is changes in the peripheral blood.

Dr. Vilter: I remember that patient quite well. She had diagnostic signs in the peripheral blood, although, just as in this instance, she did not have very severe anemia. Her red count was around three million. She had macrocytosis and a diagnostic bone marrow picture. I don't believe that one can have a diagnostic bone marrow picture without anemia in the peripheral blood. I believe that the diagnosis of pernicious anemia at least can be suspected at all times from the peripheral blood and confirmed by the bone marrow. I don't believe there are instances where no anemia occurs and yet one finds megaloblastic maturation arrest in the bone marrow. One always will have some degree of anemia. The reason I think I can say that with a fair degree of certainty is that one can take a patient with pernicious anemia in relapse, transfuse that patient up to four million red cells, and completely obliterate all evidence of megaloblastic maturation arrest in the bone marrow. I don't believe that in the natural development of the disease, one will find neurologic manifestations occurring very long before some evidence of abnormality will appear in the blood.

Dr. Aring: I certainly agree; I suppose then we must blame such statements upon the ataxia with which the blood was counted.

Dr. Vilter: I have brought to this conference the article[1] from which I believe most of the statements stem about combined system disease without pernicious anemia. I carefully went over each of the case reports therein, and I can make a different diagnosis in each instance in these patients. I can't argue with the authors, however, because in no place in the article do they say these patients have pernicious anemia nor do they say that they are sure that they are in any way related to pernicious anemia. They leave the question up in the air in the final paragraph, so I think most of the quotations in the literature are of this article and yet the article doesn't actually say what the literature says it says.

Dr. Aring: It is a tribute to your diagnostic acumen, Dr. Vilter, that you are able to make a different diagnosis in these cases because I happen to know that those that have come to autopsy to date have had multiple sclerosis.

Dr. Vilter: Well, their first case without a doubt was multiple sclerosis. It is interesting also to note that the authors state that this first case improved for a period of 10 days while getting 10 cc. of a liver extract which at that time was said to be equivalent to 50 grams of liver. Now that is not enough liver extract to do anything to a patient with postero-lateral column degeneration, nor would a patient with postero-lateral degeneration possibly improve that rapidly. What was actually happening to her was that she was going through a remission in

multiple sclerosis. She stopped the liver extract of her own volition and went for a year without it without any progression of the disease. Again this would not likely occur if it were due to pernicious anemia. She didn't have enough treatment to produce a remission to begin with, and the relapse would have occurred before a year with the small amount of liver extract that had been given her.

Patients should not die of pernicious anemia any longer. The only reason that happens is because the patient comes in too late and may have severe infection from a cord bladder, so severe that the antibiotics will not take care of the situation. They develop sepsis and die. Others die because an "eager-beaver" intern thinks that a lot of blood is just as good as a little. The house officer says, "Let's give this patient a half unit or one unit of blood." Let's say the red count is below a million and the patient is in a wobbly condition, and he thinks perhaps a little transfusion will help. The intern feels that if one unit is good, two are a lot better, and that is the quickest way to kill a patient with pernicious anemia. Either they go into pulmonary edema and die, or they go into an irreversible shock syndrome for reasons which are not too clear, but both things happen if you put in too much blood. A half unit is usually enough to tide these people over until they can get a response later from specific therapy.

Student: What would you give as the mechanism of death in this patient?

Dr. Vilter: I think the mechanism of death here was vascular collapse due to involvement from pernicious anemia of the centers in the medulla which maintain vasomotor tone. The other possibility, of course, is that she had a pulmonary infarct, but the description in the protocol doesn't suggest that.

Discussion of Pathology

Dr. Murton R. Shaver: Apparently my task is to give reason for certain of the clinical findings; Dr. Vilter's diagnosis is obviously correct. The autopsy was performed 6 hours after death. Generalized muscle wasting was obvious. Significant abnormalities included the flattened and almost totally obliterated rugae of the stomach. Some longitudinal ridges were present. The stomach mucosa was smooth, yellowish-pink; no areas of ulceration or hemorrhage were seen. The duodenum was bile stained and the ampulla of Vater could be visualized. The mucosa of the colon was grossly abnormal. This mucosa was thickened; there were raised areas of granulomatous thickening alternating with ulcerations which extended into the muscularis. There did not appear to be any associated hemorrhage or diverticulae. The raised granulomatous lesions were reddish-yellow and on the surface stained a greenish-brown. The shallow ulcers varied in size from 1 to 3 cm., they were ovoid in shape, running longitudinally in the bowel.

There was pronounced increase in cellularity in the bone marrow due chiefly to myeloid hyperplasia.

The brain weighed 1025 grams. The leptomeninges generally showed slight thickening. There was slight widening of the sulci. The vessels of the circle of Willis showed only mild atherosclerotic change. There was no evidence of a pressure cone or of any herniation of brain surfaces.

Sections of the cerebral cortex studied microscopically revealed areas of vacuolization in the white matter with little in the way of inflammatory cell infiltration and no astrocytic overgrowth. Patchy areas of demyelination accompanied the vacuolization. These degenerating areas in the hemispheres may help to explain the mental symptoms. Sections from the basal ganglia, stained to show myelin sheaths, revealed similar changes, in fact these small areas of change were scattered throughout the white matter of the brain as well as in the spinal cord. I want particularly to note the patchy areas of vacuolization and demyelination in the brain stem which may account for the bulbar signs. You may recall that the patient was in a respirator for several days prior to death.

Grossly the posterior and lateral columns of the spinal cord were pale. Microscopically there was found the typical vacuolization throughout the white matter, least in the anterior columns. I might point out that there was no increase in cellularity in

those demyelinated regions, hence the term "status spongiosus" which is characteristic of this condition, a swiss cheese appearance of the entire white matter. Glial replacement sometimes occurs after treatment with liver extract.

Myelin sheath stains revealed the great loss of myelin sheaths and the vacuolization in white matter. Bielschowsky neurofibrillar stain demonstrated the loss of axones in the white matter of the cord. There was swelling of the axones and of myelin sheaths of the roots and nerves.

Clinical Diagnosis

Pernicious anemia.

Subacute combined degeneration of the spinal cord.

Dr. Vilter's Diagnosis

Pernicious anemia.

Subacute combined degeneration of the spinal cord.

Anatomical Diagnosis

Pernicious anemia.

Subacute combined degeneration of the spinal cord with scattered vacuolization of the white matter of the brain stem and cerebrum.

Peripheral neuritis.

Atrophic gastritis.

Ulcerative colitis.

Student: I don't understand why Dr. Vilter favored failure of the respiratory center rather than intercostal neuritis.

Dr. Vilter: At first she had moderate respiratory distress, therefore, she was placed in a respirator where she remained for two days. Possibly that episode may have been due to intercostal neuropathy. Then toward the end, on the morning of the 26th, she suddenly went into shock for no apparent reason, and no mention was made of the breathing. This behavior sounded to me like that I would have expected if vasomotor control were suddenly abolished. I believe lesions that Dr. Shaver showed us in the medulla might well have finally interfered with vasomotor control. When the blood pressure drops for no apparent reason one may suspect pulmonary infarct or something of that nature; it is more likely than not to represent a vasomotor change.

Student: Do you believe that alcoholism and atrophic gastritis predisposes to pernicious anemia?

Dr. Vilter: No.

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Book Reviews

HEMIFACIAL SPASM, A CLINICAL AND PATHOPHYSIOLOGICAL STUDY. By *Robert Wartenberg, M.D.* Foreword by Macdonald Critchley, M.D. New York, Oxford University Press, 1952. x, 86 p. $4.00.

This monograph deals particularly with hemifacial spasm of the essential type, which the author prefers to call "cryptogenic." The distinctive characteristics of the disorder and the points of differential diagnosis are discussed and illustrated with full-page pictures. The close relationship of postparalytic and cryptogenic facial spasm is highlighted and the hyperkinesis in both likened to that seen in a bilateral pyramidal lesion. The author is of the opinion that reflex stimulation of the facial nerve is not the cause of the hemifacial spasm. More probably, an organic affection of the nervous system is involved, and the lesion could be infectious or degenerative in origin, or just an inferior *anlage*.

On the basis of long study of the phenomenon, the author is convinced that the clinical features implicate the facial nucleus. The spasm would be presumably a release expression of the removal of inhibition normally exercised by a structure within the facial nucleus. As in the cortico-spinoperipheral motor system, the facial nucleus must contain two elements, an upper and a lower motor mechanism. The lower system would produce hyperkinesias, the upper would be the restricting element, and if damaged, would fail to accomplish its function. The author advarnces the hypothesis that the lower motor neuron of the facial nucleus is older and stronger than the upper neuron and that the latter would therefore succumb first to damaging influences. When the facial nucleus is affected only indirectly, after injury to the peripheral branches of the facial nerve, there may be a resulting remote reaction adversely affecting the younger part of the nucleus.

This little book is admirably conceived, presented, and documented. It offers a reasonable new approach to an old and puzzling problem in clinical neurology.

PROJECTIVE TEST PRODUCTIONS. I. PROJECTIVE DRAWINGS. By *Claire Myers Vernier, Ph.D.,* Chief Clinical Psychologist, V. A. Center, Martinsburg, W. Va., Foreword by Isabelle V. Kendig, Ph.D., Chief Psychologist, V. A. Hospital, Tomah, Wis. New York, Grune & Stratton, 1952. vii, 168 p. $6.00.

The author presents a sampling of projective drawings created by patients suffering from various types of psychiatric disorder as well as by a group of normal individuals. The cases selected were clear-cut examples of a particular diagnostic group according to the independent judgment of psychiatrists and other medical specialists.

In the projective drawing test, the subject is asked to sketch a male and a female person. Experience has indicated that the creation of the patient may prove useful in eliciting overall concepts of self and opposite sex, since there is a frequent tendency for the patient to identify with the figures drawn. However, the author is in agreement with many other workers that projective drawings are not primarily a diagnostic test and should be used only as one of a battery. The material presented in this book shows a certain overlap of so-called abnormal features, both in the psychiatric groups and in the normal series.

The following groups are represented in the sampling: schizophrenia; manic-depressive psychoses; involutional psychoses; cases of organic pathology; mental deficiency; other types of psychoses, including alcoholic and psychopathic cases; nonpsychotic cases of psychopathy and addiction; cases with psychosomatic features, without psychosis; psychoneuroses; and normals. A brief discussion of the general feature of the drawings in the group precedes each section, and each drawing or set of drawings is accompanied by a brief description of the outstanding features of the case, followed by comments on various aspects of the drawings. An appendix provides a summary table of data for all 60 figures incorporated in the book, and, for purposes of laboratory exercises, a series of drawings produced by five unidentified individuals.

This volume is intended as a classromm aid rather than a text in the interpretation of projective drawings. As such, it should prove useful and popular.

PROBLEMS OF CONSCIOUSNESS. Transactions of the Second Conference, March 19-20, 1951, New York, N. Y. Edited by *Harold A. Abrahamson, M.D.* Sponsored by the Josiah Macy, Jr. Foundation. Josiah Macy, Jr. Foundation Publications, Packanack Lake, N. J., 1951. 178 p. $3.25.

Incorporated in this volume are the Transactions of the Second Conference on Problems of Consciousness, which was attended by a group of 24 eminent authorities in medical, social and allied sciences. Among the disciplines represented, in addition to psychiatry and neurology, were electroencephalography, physiology, anatomy, biology, zoology, anthropology, psychology, and philosophy. The diversity of interest and methodology thus symbolized enriches the discussion and the conclusions reached at the Conference.

The program was organized around three topics prepared respectively for the meeting by David Rapaport, Gregory Zilboorg, and David G. Wright. Dr. Rapaport gave a psychopathological and psychodynamic view of consciousness, emphasizing formal characteristics of conscious experience and the effect of emotional needs on the functions of consciousness. Dr. Zilboorg spoke about variations in the scope of awareness, with particular reference to the content of conscious experience and its impact upon the individual. Dr. Wright, using the case study approach, described variations in states of awareness in schizophrenic patients.

These three reports are of great interest in themselves, but the major appeal of the book lies in the multifaceted, scintillating discussion reported verbatim. The publication shows how convergent are the problems of an heterogeneous group of this kind, and points up the need and feasibility of the communication and integration of data and ideas.

A TEXTBOOK OF CLINICAL NEUROLOGY, with an Introduction to the History of Neurology. By *Israel S. Wechsler, M.D.* Seventh Edition. Philadelphia and London, W. B. Saunders Company, 1952. xiv, 801 p.

In the seventh edition of his textbook, Wechsler has incorporated the most recent advances in clinical neurology, deleted obsolete material, and revised the text generally in the light of new experience.

There is little change in the organization of subject matter. The author presents signs and symptoms of the various diseases, their development and their course, in the light of the anatomico-pathological substrate and the underlying physiological disturbance, and illustrates the text largely with pathological specimens and anatomical drawings.

One notes a thorough revision of sections dealing with the meningitis, especially tuberculous meningitis and other infections of the nervous system, and a new emphasis on the diagnostic use of angiography. Neurosyphilis, injuries of the brain and spinal cord, the surgery of tumors have been carefully reviewed, and electromyography and double simultaneous stimulation are newly incorporated.

This textbook has been very popular in the twenty-five years of its existence. Frequent revisions make it a useful source of reference. It contains a chapter on psychological diagnosis, by Dr. David Wechsler, a comprehensive survey of the neuroses, and a chapter on the history of neurology.

DYNAMIC PSYCHIATRY. Edited by *Franz Alexander, M. D.,* and *Helen Ross.* Chicago, The University of Chicago Press, 1952. xii, 578 p. $10.00.

The main emphasis of this volume is the mutual interactions of psychoanalysis with allied fields. It is a work of multiple authorship, well planned and edited, and notably documented. The contributors, fifteen in number, are experts in their areas of discussion.

The material is organized into three parts. The first deals with the basic concepts of dynamic psychiatry and includes contributions by Franz Alexander, Thomas M. French, Edoardo Weiss, and Therese Benedek. The second is concerned with the application of those concepts to clinical psychiatry, with emphasis on neurotic reactions, child psychiatry, organic cerebral disorders, psychoses, and, finally, principles of psychiatric treatment. The contributors include Franz Alexander and Louis B. Shapiro, Leon J. Saul and John W. Lyons, Margaret W. Gerard, Henry W. Brosin, John C. Whitehorn, and Maurice Levine. The third part deals with the influence of psychoanalysis on such allied fields as psychosomatics, social anthropology, clinical psychology, and animal psychology, and concludes with a chapter reviewing the influence of psychoanalysis on current thought.

This volume is a distinctive addition to the literature on dynamic psychiatry. It presents fundamentals with clarity and precision and can be recommended to a wide circle of readers.

THE BATTLE FOR MENTAL HEALTH. By *James Clark Moloney, M.D.* New York, Philosophical Library, 1952. x, 105 p.

The author is an eloquent spokesman for the point of view that a warm, relaxed relationship between parents and children is basic to sound emotional development. He has made previous interesting observations on the subject in other cultures. This little book presents an elaboration of the above idea. The author paints a rather macabre picture of the contemporary American scene where he finds the number of completely normal people alarmingly small, the cultural orientation in itself destructive to mental health, and the attitude toward the American infant misguided and sado-misanthropic.

After presenting his view on the etiology of mental ill health, the author suggests a few steps for a more positive trend. He speaks of the need for less highly institutionalized maternity facilities and improved mother-baby relationships. He describes the endeavors of the Cornelian Corner in Detroit on behalf of the permissive method of child-rearing, and analyzes the types of hostility and resistance toward the work which currently prevail.

There are a number of interesting concepts in this book, despite the width of some of its statements. Physicians may be startled to hear that "most of the maternal emotional disorders in our culture can be traced back to the inferiority complexes of our physicians."

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